Southwestern MEDICINE

Official Journal of The Southwestern Medical Association, The Western Association of Railway Surgeons, The Texas Orthopaedic Association, The Southwest Obstetrical and Gynecological Society, The Southwestern Dermatological Society, Texas District One Medical Association, The Southwestern New Mexico Medical Society, and El Paso County Medical Society

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IN THIS ISSUE

The President's Page
Young Men of Medicine ..... **Page 145**

Dr. Russell Holt of El Paso Elected President of Texas District One Medical Association ..... **Page 147**

Two Speakers to be Featured at Texas Orthopaedic Meeting in Fort Worth ..... **Page 148**

Post-Graduate Psychiatric Courses for GPs Scheduled in Phoenix ..... **Page 149**

A New Treatment for Peptic Ulcer ..... **Page 150**

Clinical Pathological Conference
R. E. Thomason General Hospital, El Paso ..... **Page 159**

COMPLETE CONTENTS ON PAGE 136

March, 1960

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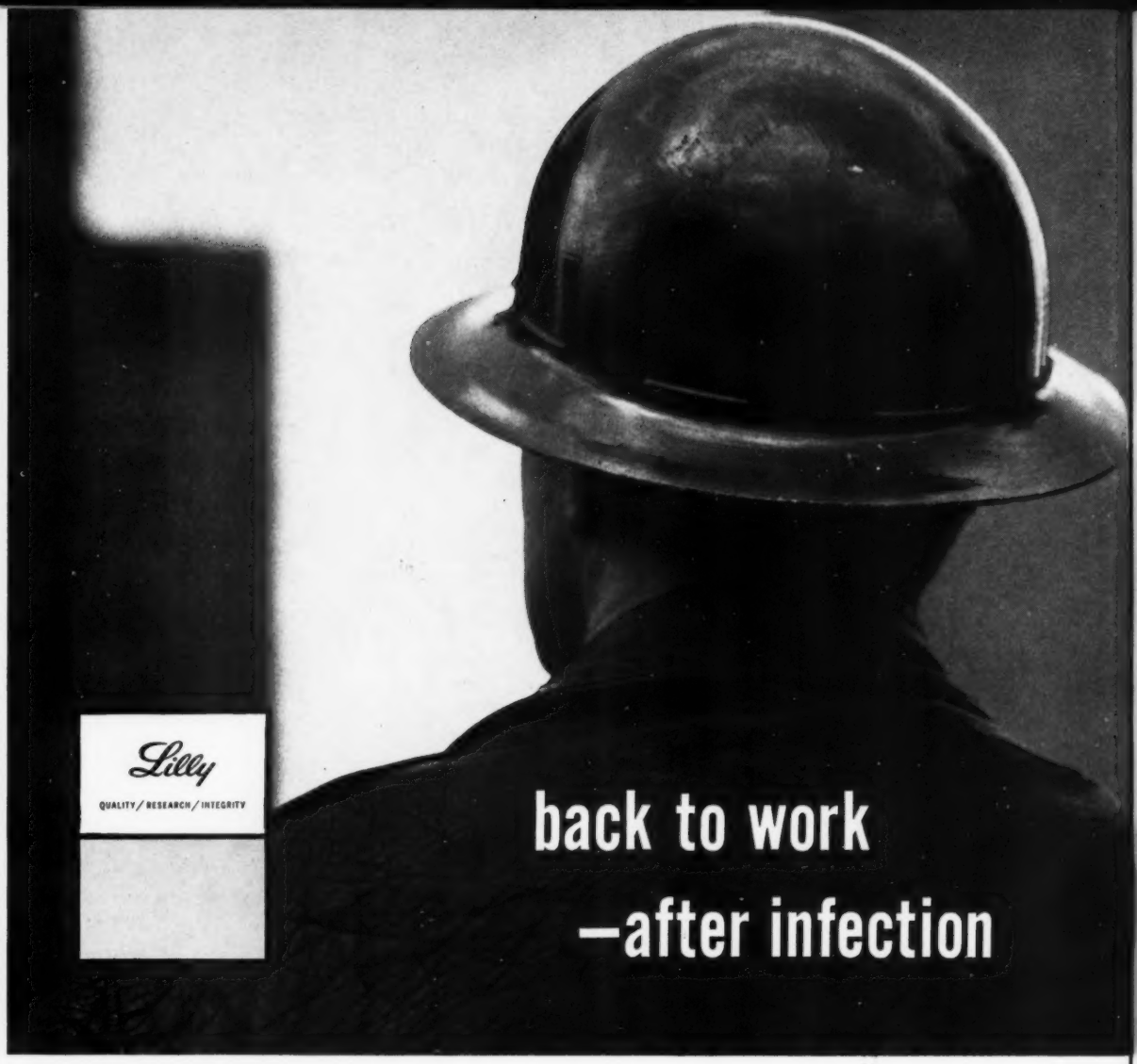

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Contents

The President's Page — Young Men of Medicine ..... Page 145
By Russell L. Deter, M.D., El Paso

Dr. Russell Holt of El Paso Elected President of Texas District One Medical Association ..... Page 147

Two Speakers to be Featured at Texas Orthopaedic Meeting in Fort Worth ..... Page 148

Post-Graduate Psychiatric Courses for GPs Scheduled in Phoenix ..... Page 149

A New Treatment for Peptic Ulcer ..... Page 150
By Alexander Coblentz, M.D., Las Vegas, Nevada

Clinical Pathological Conference, R. E. Thomason General Hospital, El Paso ..... Page 159

Frederick P. Bornstein, M.D., Editor

Presentation of Case by Charles Logsdon, M.D.

Coming Meetings

Medicine and Surgery Symposium, Hilton Hotel, El Paso, March 12, 1960, sponsored by El Paso County Medical Society and Lederle Laboratories.

American College of Surgeons, Sectional Meeting, The Broadmoore Hotel, Colorado Springs, March 21-23, 1960.

United States - Mexico Border Public Health Association, Annual Meeting, Hermosillo, Sonora, Mexico, April 4-8, 1960.

Texas Orthopaedic Association, Annual Meeting, Hilton Hotel, Fort Worth, April 11, 1960.

Arizona Medical Association, 69th Annual Meeting, Safari Hotel, Scottsdale, Arizona, May 4-7, 1960.

New Mexico Medical Society, Annual Meeting, Western Skies Hotel, Albuquerque, May 11-13, 1960.

Postgraduate Course, Dermatology for General Practitioners, University of Colorado Medical Center, Denver, July 14-16, 1960.

Postgraduate Medical Assembly of South Texas, Shamrock Hilton, Houston, July 18-20, 1960.

Southwestern Medical Association, 42nd Annual Meeting, Hotel Hilton, El Paso, Oct. 20-22, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Riviera Hotel, Las Vegas, Nev., Nov. 7-8, 1960.

Facts

GREATER INHIBITORY ACTION / LOWER DAILY MILLIGRAM INTAKE / BROAD-SPECTRUM CONTROL IN GREATER DEPTH / CONSTANT PEAK ACTIVITY / EXTRA-DAY PROTECTION AGAINST RELAPSE

Findings

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SOUTHWESTERN MEDICINE

VOL. XLI, NO. 3 MARCH, 1960

Young Men of Medicine

By RUSSELL L. DETER, M.D., *El Paso*

President, Southwestern Medical Association

NOTE: Dr. Deter will discuss medicine's responsibility to the medical student, intern and resident — and the young doctor starting out in practice — in next month's issue.

EDITOR

Dr. Deter

The most exciting thing in medicine today is, as a group, the young men of medicine. Who are the young men of medicine? There are four categories:

1. The high school student who wants to study medicine.

2. The pre-medical student in college.

3. The medical student, intern and resident.

4. The young doctor beginning his practice of medicine.

Let us take these in order: First, the high school student who wants to study medicine. It is at this level that all of us, regardless of length of time in the practice of medicine, must exercise our utmost to encourage bright young minds to become interested in the field of medicine, impress them with the variety which exists, and the lack of monotony which is present in the practice of medicine, impress on them that the only security you can get is what you work for, and earn, impress on them that when you get a bouquet from "Uncle Sam" the flowers are always gathered in your front yard!

As Secretary of the Treasury Anderson stated in a letter to a young friend: "I would give you only one word of caution. When a boy is possessed of great abilities, as I think you are, he owes, in my judgment, much more to the world than those of lesser ability. This means an obligation to study, to learn, to think soundly and wisely, to be tolerant and understanding of others, and to maintain a sense of humor and patience. The way to success for anyone is never very easy, it comes from long hours of hard work that the rest of the world will little note nor long remember. I am sure that you are probably aware of these things already, but I know that I keep reminding myself that they are part of life's essentials."

Desire

Look for honesty, integrity and inquisitive mind — *desire*. One could write an entire treatise on the one word. You have got to *want to* to succeed. The same is true now as it was one hundred years ago.

2. The college student. The job here is much the same, but if you have done your job well at the high school level, here it would be much simpler. Here your job is one of advising him on how to prepare himself for medical school. Impress upon him the importance of being a well educated, well rounded citizen of his community. Also, that it is at the college level that he must

THE PRESIDENT'S PAGE

learn something about city, county, state and Federal government and his responsible place in it. That, as a physician, he must not only treat the physical ills of his community, but the social ills also. He must be impressed that a great amount of his time will be taken from making a living to participating in community activities. That he should select projects close to his heart and interest, and back them financially as well as physically — such as the whole gamut of youth programs — YMCA, Boy Scouts — even the care of the indigent can come under this heading.

They *do not know* these things, coming into medicine stone cold. How much easier it would be to interest doctors as a whole, if all of them could have been so prepared as they come along. Impress upon them the importance of knowing the English language, public speaking, writing, the humanities, philosophy, sociology, government, music appreciation, art, ad infinitum. Preparation for medical school does not involve taking courses that will be repeated in greater detail in medical school. One only wastes that time that could be used to greater advantage in taking those courses for which there is not sufficient time in medical school.

Through all of this, and at all levels, do not leave out religion. There is nothing that gives a man humility, no matter what his success, like being close to God.

Youth is such a wonderful age. They have never been able to realize that they are on the brink of disaster, as the gloom seekers would want them to believe. Because of this wonderful endowment of youth, each generation succeeds where the last one failed.

AT PECOS MEETING—Participating in the District One meeting of the Texas Medical Association in Pecos, Texas, February 5, 1960, were, left to right, Dr. Charles Oswalt, Fort Stockton, Councilor for District One and Chairman of the TMA Board of Councilors; Dr. Franklin W. Yeager, Corpus Christi, President of the Texas Medical Association; and Dr. William Lockhart, Alpine, immediate Past President.

MEETINGS

Dr. Holt

Dr. Russell Holt of El Paso Elected President Of Texas District One Medical Association

Dr. Russell Holt of El Paso was elected President of District One of the Texas Medical Association at its annual meeting in Pecos, February 5, 1960.

Other new officers are Dr. Harold Lindley, Pecos, Vice-President; Dr. Gordon L. Black, Secretary-Treasurer; Mrs. Louis W. Breck, El Paso, Councilwoman for the District One Auxiliary. Dr. W. E. Lockhart was the retiring President.

Dr. Franklin W. Yeager of Corpus Christi, President of the Texas Medical Association, cited the need for more family physicians. He said that medical societies should inform the public about the availability of their grievance committees.

Creeping Socialism

He said that if creeping socialism were to take over the medical field, it would next embrace insurance, then banking and construction, power companies and finally achieve a complete socialism.

Born in Palestine, Texas, Dr. Holt received his B.A. from Texas Tech and his M.D. from Colorado University Medical School. He interned and did residency work at Robert B. Greene Hospital in San Antonio. He came to El Paso in 1934 as a general surgeon.

Past President

He is a Past President of the El Paso County Medical Society and the Lions Club. He has served as Chief of Staff at Hotel Dieu and the old City-County Hospital in El Paso, and is a member of the Hospital District Board. He is a member of the First Christian Church. He has a daughter, Mrs. E. S. Holmes, who resides at Fort Sill, Okla.

Drs. E. S. Crossett and John M. Verosky of El Paso announced at the meeting that El Paso has been selected as a regional center for the heart division of the Crippled Children's program.

Other speakers were Drs. Ira A. Budwig, C. M. Stanfill, Jack C. Postlewaite, Nathan M. Kleban, William G. Smith, Albert H. Unger, Louis W. Breck and Frederick P. Bornstein.

Dr. and Mrs. Harold Lindley of Pecos were in charge of arrangements for the meeting.

Two Speakers to Be Featured

At Texas Orthopaedic Meeting

Dr. Don O'Donoghue of Oklahoma City and Dr. W. H. Bickel of Rochester, Minnesota, will be featured speakers at the annual meeting of the Texas Orthopaedic Association in Fort Worth April 11, 1960.

Dr. O'Donoghue is Professor and Chairman of the Department of Orthopaedic Surgery and Fractures at the University of Oklahoma Medical School. Dr. Bickel is Consultant in Section of Orthopaedic Surgery at the Mayo Clinic and Professor of Orthopaedic Surgery for the Mayo Foundation, Graduate School, University of Minnesota.

The meeting will be held in the Hilton Hotel in conjunction with the annual gathering of the Texas Medical Association in Fort Worth. The orthopaedic meeting will be in the New Orleans and Derrick Rooms of the Hilton Hotel with a luncheon in the Empire Club.

Other speakers will be Dr. Phil Overton, Jr., Dallas; Dr. P. M. Girard, Dallas; Dr. E. Burke Evans, Galveston; Dr. George W. N. Eggers, Galveston; Dr. David M. Cameron, El Paso; Dr. Royce C. Lewis, Jr., Lubbock; Dr. Louis J. Levy, Fort Worth; and Dr. Bruce Cameron, Houston.

Officers Listed

Officers of the Association are Dr. David M. Cameron, El Paso, President; Dr. Gerald S. Ahern, Corpus Christi, Vice-President; and Dr. Margaret Watkins, Dallas, Secretary-Treasurer. Program Chairman for the meeting is Dr. Rex J. Howard, Fort Worth.

The complete program follows:

9:30- 9:50 "Experimental Fetal Bone Transplant"

Dr. Phil Overton, Jr., Dallas

Dr. O'Donoghue

Dr. Bickel

9:50- 9:55 Discussion
Dr. Charles Gregory, Dallas

10:00-10:20 "Dual-Lock Hip Prosthesis"
Dr. P. M. Girard, Dallas

10:20-10:25 Discussion
Dr. Russ B. Graham, Dallas
Dr. A. O. Loiselle, Dallas

10:25-10:30 INTERMISSION — Coffee served

10:30-10:50 "Two-Plane Fixation of Femoral Fractures"
Dr. E. Burke Evans, Galveston
Dr. George W. N. Eggers, Galveston.

10:50-10:55 Discussion
Dr. Louis Breck, El Paso

11:00-11:40 "Surgical Treatment of Ligament Injuries To The Knee"
Dr. Don O'Donoghue, Oklahoma City

11:40-12:00 Discussion
Dr. Louis J. Levy, Fort Worth

12:00 2:00 LUNCHEON, EMPIRE CLUB

2:00 2:25 President's Address: "Orthopaedic Surgery — An Art"
Dr. David Cameron, El Paso

2:30 2:50 "Management of Crush Injuries of The Hand"
Dr. Royce C. Lewis, Jr., Lubbock, Texas

2:50- 2:55 Discussion
Dr. Robert Cochran, Fort Worth

3:00- 3:20 "Follow-up Evaluation of Fractured Heels"
Dr. Louis J. Levy, Fort Worth

3:20- 3:25 Discussion
Dr. Jack Maxfield, Wichita Falls, Texas

3:30- 3:50 "Study of the Biomechanics of Whiplash Injuries of the Neck"
Dr. Bruce Cameron, Houston

3:50- 3:55 Discussion
Dr. Ruth Jackson, Dallas
Dr. George W. N. Eggers, Galveston

4:00- 4:40 "Postirradiation Fractures of the Neck of the Femur"
Dr. W. H. Bickel, Rochester, Minnesota

4:40- 5:00 Discussion
Dr. Robert Murray, Temple, Texas

Post-Graduate Psychiatric Courses For GPs Scheduled in Phoenix

A series of post-graduate seminars for nonpsychiatrist physicians in Arizona is scheduled to begin in March, 1960, in Phoenix and will consist of two-hour seminars held one evening each week for ten consecutive weeks. The course is designed for physicians who practice in communities which are isolated from medical training centers, as an aid to nonpsychiatrist physicians in recognizing and understanding psychiatric problems. The program will not stress the management of psychiatric problems per se. Focus will be on doctor-patient relationships and on professional relationships between psychiatrists and non-psychiatrist physicians.

Case material will be provided by the physicians themselves from their current practices. Sessions will be guided by two eminent psychiatrist-teachers from Phoenix. As an integral part of the course, the psychiatrist-teachers will be available for telephone consultations with the physician-trainees between seminar meetings. Time and place of the weekly meetings will be determined by the teachers and the trainees. Any qualified physician is eligible to apply for enrollment in the ten-week course. Course enrollment will be limited to fifteen physicians. These seminars will be the first in a three-year program and will be conducted simultaneously in Billings, Montana; Lebanon, Oregon; and Phoenix, Arizona.

Training Program

This training program for nonpsychiatrist physicians was developed by the Western Interstate Commission for Higher Education and is supported by congressional funds through the National Institute for Mental Health. The WICHE staff is responsible for all administrative details related to the program. Warren T. Vaughan, Jr., M. D., Boulder, Colorado, is the Mental Health Project Director for WICHE. A detailed report will be published after the three-year program has terminated. In Arizona, the program is directed by the Arizona State Medical Association, Inc., Robert Carpenter, Executive Secretary.

ORIGINAL ARTICLES

A New Treatment For Peptic Ulcer

By Alexander Coblentz, M.D., *M. Med. Sc. (Surgery), Las Vegas, Nevada*

This preliminary report is made in order to draw attention to the value of a new treatment for the patient with an acute bleeding or non-bleeding gastric or duodenal ulcer, by the combined use of oral *Cosa-Signemycin with intramuscular chymotrypsin.**

This investigation was carried out at the Southern Nevada Memorial Hospital, department of surgery with the cooperation of the roentgenology department.

In ten consecutive cases; eight males and two females, ages 23 to 64, there was a peptic ulcer with either active hemorrhage, severe discomfort or a sealed-over perforation. In two cases, surgical intervention was contraindicated owing to the patients' desperate physical condition. One patient, on exploratory laparotomy, was found to have a sealed-over perforation of the first portion of the duodenum. Seven of the patients had a hematocrit ranging from 4.1 to 9.4. Three of the patients showed no evidence of gastric or duodenal bleeding.

Chart 1 contains the data of the cases in this series before and after treatment.

The admitting information, history, physical examination, roentgenological and laboratory findings were conclusive for a gastric or duodenal ulcer.

Following the prescribed 10 day course of treatment, as described in Chart 2, there were no roentgenological or other evidence of an ulcer of the stomach or duodenum, see Chart 1.

During recent years the indications for surgical intervention for hemorrhage in cases with peptic ulcers, have been clearly defined. However, occasionally surgical intervention is contraindicated owing to the patient's desperate physical condition (1).

Excluding those ulcers associated with lesions of the hypothalamus or extensive burns, the majority bear no relationship to other disease processes. In all likelihood they have a similar etiology.

All etiological theories pertaining to gastric or duodenal ulcers are inadequate. Nevertheless, if a a nursing calf is taken too soon from its mother and given coarse foods, it is prone to develop erosions due evidently to mechanical irritation (2). It is probable that in man the common cause of weakened resistance which gives rise to peptic ulcer, may also be a local irritation, followed by infection of the local erosion.

Acute Stage

The ulcer in the acute stage has a punched-out appearance with soft congested walls and floor, showing a typical inflammatory reaction. Healing of the ulcer may occur without serious change, the remnants being a smooth scar.

The chief abnormality in ulcer patients is the secretion of large amounts of gastric juice in the intervals between meals, particularly at night when the stomach is empty.

Whether the precipitating factors are gastric trauma, local infection, nervous tension or hyperacidity due to hypersecretion, the main factors are the hypersecretion and hyperacidity which contains the protein digestive enzymes — pepsin and hydrochloric acid. They convert the area of pre-ulcer necrosis into an actual ulcer.

In reviewing the physiological chemistry of the gastroduodenal ulcer, consideration was directed to the method of stimulation of the gastric enzymes; also to the action of the enzymes in producing an active ulcer from the original area of erosion within the stomach or duodenum, and the specific type tissue digested by the enzymes pepsin and hydrochloric acid.

Noteworthy Property

One of the noteworthy properties of enzymes is their tissue specificity. This is exhibited as absolute specificity, i.e. pepsin will digest proteins, not fat

*Product of Pfizer Laboratories.

**Chymar, chymotrypsin (aqueous or in sesame oil suspension) a product of The Armour Laboratories, Kankakee, Illinois.

Case No.	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Hospital No.	55,310	57,631	60,946	12,459	63,048	63,255	12,976	63,641	63,696	63,847
Initials	L.R.	H.H.	T.R.S.	J.L.	G.G.R.	E.L.B.	N.C.	L.T.P.	F.I.	H.H.G.
Sex & Age	M 46	M 47	M 42	M 33	M 40	M 64	F 27	M 51	F 46	M 54
Occupation	Dept. Stor	Dealer	Dealer	Dealer	Construc'n	City Emp.	Hw	Laborer	Hw	Dealer
Chief Complaint on Admission	Vomiting Pain Bleeding	Pain Hem'ge Vomiting	Weakness Dyspepsia Melena	Pain Melena	Vomiting Pain Bleeding	Melena Vomiting Bleeding	Pain Dysp' Vomiting	Pain Anorexia Dyspepsia	Pain Anorexia Dyspepsia	Vomiting due to obstruc'n
Past Ulcer History	7 years	8 months	7 years	3 years	12 years	2 years	None	None	3 years	4 years
Temperature Admission (oral)	99.F	98.0	100.2	N.D.	99.6	100.6	98.0	101.2	98.2	97.6
Hematemesis	++++	++++	++++	++++	++++	++++	None	None	None	None
Melena	++++	++++	++++	++++	++++	++++	None	++++	None	None
Blood Group	A	O	A	A	AB	O	O	O	O	O
Admission Hemoglobin gms	4.15	8.6	5.35	9.4	9.1	8.85	N.D.	9.0	13.6	13.75
Admission Hematocrit Vol%	13	25	19	N.D.	28	27	N.D.	28	40	43
X-ray Diagnosis Date	Duodenal Ac. Hem'g 8-5-58	same 11-7-58	same 4-27-59	same 6-19-59	same 8-17-59	same 8-25-59	Duodenal Acute 9-18-59	Duod.** Ac. Peptic 9-22-59	Duod. Acute 9-23-59	Obstrc'n*** Ac. Duod. 9-28-59
Follow-up X-ray Results Date	Healed 9-25-58	Healed 1-12-59	Healed 5-11-59	Healed 7-16-59	Healed 8-31-59	Healed 9-15-59	Healed 10-9-59	Healed 10-5-59	Healed 10-7-59	Healed 10-13-59

CHART 1

N.D. — Not Done

** — Perforated

*** — Ulcer with psuedo diverticulum

#Dealer — Gambling Casino

or carbohydrates. Dipeptidase hydrolyzes only dipeptides and not the higher polypeptides (3, 4, 5, 6, 7).

The tissue involved in a gastric or duodenal ulcer is a protein.

Some intestinal worms resist the enzymatic action of the gastric juices. For example, the intestinal worm Ascaria escapes digestion in the human intestinal tract because the worm contains a substance which inhibits pepsin (3, 7, 8, 26).

Although this system is very complicated, the introduction of substances into the body can stimulate the existing inhibitors in the blood stream. These inhibitors can be of several different types, first those that negate proteolytic activity, and the second those that interfere with one or another stage of the activation of the enzyme.

Pepsin inhibitors, which appear to be proteins, are found in pancreas, soy bean and raw egg white (3, 9). It is known that animals can be made to react to chemicals as well as to foreign proteins and other animal enzymes. These substances causing enzyme inhibition effects.

An example of this effect is the sulfonamide derivative acetylzoleamide which, although inactive as a bacteriostatic agent, is a potent inhibitor of the activity of the enzyme carbonic anhydrase (3), an enzyme found in erythrocytes and other tissues and which catalyzes $H_2O + CO_2 \rightleftarrows H_2CO_3$. Acetylzoleamide (Diamox) has been found applicable in the control of water and electrolytes excretion because of the importance of carbonic anhydrase in the reabsorption activity of the renal tubules.

Mammalian pancreas contains a protein conjugate chymotrypsin; and this enzyme when injected into the human causes sythesis of protein-like substances through its biological activity, causing transpeptidation reactions negating peptic activity (16).

When given parenterally to patients with gastric or duodenal ulcers, chymotrypsin acts as an enzyme inhibitor, which neutralizes and inhibits secretion of hydrochloric acid with simultaneous elimination of peptic activity in the gastric juice and duodenal mucus (20, 21, 22).

CHART 2

1. Medication

—Blood and fluid replacement as indicated by laboratory findings
—Chymotrypsin* 1 cc parenterally q 12 hours
—Cosa-signemycin 250 mg orally q 6 h
—Antacid tablets or liquid at bedside for P R N use
—In case of hemorrhage Vitamin K 75 mg intramuscularly q 6 h until bleeding is controlled
—Codeine Sulfate grains i (H) q 6 h when indicated
—Tranquilizers for apprehensive cases
—High potency vitamins

2. Diet

—The diet is arranged to gradually increase the kinds and amounts of foods. It is started with a soft diet, with the addition of cooked cereals and half and half cream between meals. When improvement is recognized a regular diet (with the exceptions as indicated below) is started, usually on third day. Excluded are uncooked fruits and vegetables and fruit juices.

3. Not Allowed

—Alcohol, tobacco, vinegar, salad dressings, spices, condiments, prepared, salted or smoked, fatty or fried meats and fish; whole grain cereals, pies, pastries and concentrated sweets. Avoid hot foods, and pork products.

With the combined use of Cosa-Signemycin to control the cellulitis within the ulcer process, prompt relief from pain is usually affected within 72 hours. Following ten days of treatment there is complete absence of ulcer symptoms. Roentgenological evidence of complete healing may be seen as early as two weeks following the start of treatment.

One serious complication of "cured" duodenal ulcer treated by methods such as alkalinization, lavage, etc., is the development of scar tissue capable of producing pyloric obstruction, or a stricture of the duodenum itself. None of the cases treated with the combined Cosa-Signemycin/chymotrypsin method have shown any evidence of scar tissue formation in the follow-up X-ray examination.

All the patients tolerated the medication well, no nausea and vomiting or other side effects were noted. The use of chymotrypsin and Cosa-Signemycin does not seem to produce any deletorious effects on the patient. The over-all impression was that patients on therapy with this enzyme and antibiotic were remarkably free of discomfort. There were no undesirable reactions.

The following cases taken from Chart 1 are the more serious types of peptic ulcer cases and are excellent examples of the effectiveness of this treatment.

Case 1, L.R., Hospital #55310, male, age 45, was readmitted to the hospital on 7-27-58 with a history of sudden severe hematemesis and copious tarry stool. This attack was preceded by anorexia, vague epigastric distress, marked nervousness and dyspepsia, requiring antacids. The past history revealed hospitalization for upper G-I bleeding due to duodenal ulcer on 11-15-51, 12-14-52, 12-21-52, 7-14-53, 6-19-56.

Stormy Course

His condition following admission to the hospital followed a stormy course. The bleeding became severe and on 7-31-58 the hemoglobin was 27.8% and 4.14 grams, with a hematocrit of 16%. He was placed on a Sippy diet #1, Demerol 100 mg P R N., Synkovite 75 mg q 6 h, Streptomycin with Chlormycetin grams 1 each q 6 h, and Whole Banked Blood, 5 units.

On 8-2-59 the author was called in as consultant.

Following a thorough evaluation of the patient, the patient was considered in too desperate physical condition to withstand surgery. The hemoglobin on 8-2-58 was 4.15 grams, hematocrit was 13 volume %, melena was marked.

Due to his critical condition, he was placed on the treatment as described in Chart 2. His response was gratifying, and on 8-5-59 the patient was examined roentgenologically. The film studies confirmed the subjective and objective findings, revealing a rather large, irregular deep or penetrating ulcer on the posterior aspect of the bulb near the apex. The treatment with Cosa-Signemycin and chymotrypsin was continued to complete the ten day course of treatment. His progress was excellent and on 9-25-59 the X-ray reexamination report was as follows: the gastric mucosa motility and peristalsis were within normal limits.

The duodenum was reexamined rather exhaustively with special reference to the area previously reported as the site of an ulcer. The bulb filled and emptied well, was not spastic or tender. There was a persistent mucosal irregularity near the apex in the bulb, rather shallow in contour, which may represent a residua from the previously reported peptic ulcer. There is no current evidence of hypersecretion, no evidence of pyloric spasm or stenosis of this region or the bulb, no marked psuedo-diverticulum to suggest the possibility of impending perforation. The proximal small bowel mucosa visualized was within normal limits, and the barium was in the terminal ileum within three hours with minor minimal quantities in the colon. Impressions: Current radiographic findings are

compatible with a healing or healed duodenal ulcer, with no current evidence of spasm, hypersecretion, or complications.

General Weakness

Case 3, T.R.S., Hospital #60,946, male, age 42. admitted 4-25-59, for what the patient believed was an acute episode of influenza, due to generalized weakness and frequent loose black stools for the past ten days. The past history shows that he had been under treatment for a duodenal ulcer several years ago. He had been obtaining relief from soda mint tablets, milk of magnesia, ordinary baking soda in water, Aspirin and Aspirin containing preparations. Physical examination showed the patient to be well developed, anemic in appearance, no evidence of vitamin deficiency. Liver and spleen not palpable nor tender. There was moderate tenderness of the epigastric region with no other positive findings. Laboratory findings were WBC 5,300, RBC 1,530,000 with Polys 67%, lymphs 23, Mono 9. The Hemoglobin 34.5%, 5.35 gms, Ht 19 Vol. %. Urine was negative. Stool black and contained blood. He was given 2 units WBB, plus 2,000 cc gastric isolytes.

The X-ray examination on 4-27-59 showed there was no abnormality of the esophagus, no cardia spasm, no hiatus hernia, no varices. The stomach was empty at the beginning of the examination. The mucosal pattern was within normal limits; there was no tenderness to palpation, mobility was normal. The pylorus was adequately patent. The duodenal bulb was markedly spastic but not remarkably tender. Multiple spot films show hypertrophy of the mucosa, irregularities compatible with scar tissue contraction and one or more small areas compatible in appearance with peptic ulcer. Due to the capacity of the bulb, the location of these defects in the anterior or the posterior wall could not be made with certainty. The duodenal loop was normal in appearance. The proximal small bowel mucosal pattern was unremarkable.

Follow-up Films

A series of follow-up films taken for small bowel shows the stomach completely empty at thirty minutes and the proximal jejunum shows a normal mucosal pattern which is relatively well cleared. The distal jejunum and ileum showed rather marked sudden changes in caliber characteristic of hyperperistalsis but no indications of mucosal edema. The first barium that entered the colon at thirty minutes from the small bowel was essentially clear at one hour and one-half with only traces remaining at three hours. At this time the barium column was at the spenic flexure. Impressions: Chronic duodenal ulcer disease, probably active with multiple small areas compatible in appearance with acute peptic ulcer; hyperperistalsis and segmentation with a remarkably increased transit time through the small bowel. This may be neurogenic in origin or be a manifestation of bleeding.

Therapy with Cosa-Signemycin/chymotrypsin was started immediately following the latter report. The immediate effect upon the patient was seen in the following laboratory findings of 5-1-59: R B C 3,380,000, Hb 59.1%, 9.1 grams, Hematocrit 29 vol %. The progress of the patient was uneventful and he was discharged from the hospital. Upper G-I reexamination was done on 5-11-59 with the following report: A repeat examination again demonstrates normal function, structure and mucosal pattern of the esophagus. The stomach was not tender, mucosal pattern was normal with no evidence of ulceration. In contrast with the first examination, the duodenal bulb was no longer spastic, nor tender.

Multiple films taken with the direct and air contrast techniques showed a marked decrease in the previously noted edema of the duodenal mucosa and on this examination failed to demonstrate any indication of acute or chronic peptic ulcer. The duodenal loop was normal. The proximal small bowel was within normal limits. Transit time through the colon was somewhat accelerated but in contrast to the previous study, the mucosal pattern of bowel was much improved. Impression: No radiographic or fluoroscopic findings of a duodenal ulcer, marked improvement of the mucosal pattern and the fluoroscopic behavior of the duodenal bulb.

Hyposthenic Status

Case 8. L.T.P., Hospital #63641, 51 year male, hyposthenic status was first seen on 9-12-59, when he was seized with acute colicky pain immediately over the gall bladder region. All factors suggested passage of a gall stone. Intravenous cholangiograms obtained twenty-four hours later on 9-13-59

revealed a functioning gall bladder with what appeared to be two bands across the ampulla and neck of the gall bladder. There was no significant allergic history, no reaction to the test dose of intravenous injection of 20 cc's of 50% Cholografin.

A preliminary film showed no calcification within the biliary tree. Moderate costochondral calcification in the region was noted.

At five minutes, a scout film showed essentially no secretion of the biliary contrast medium by way of the urinary tract, an early and excellent outline of the gall bladder and right and left hepatic ducts and a portion of the common duct. Multiple films in several projections were taken over a period of the next two and one-half hours. These disclosed two permanent deformities of the gall bladder, one approximately one-third of the way through the fundus of this structure, one at the junction of the cystic duct. Appearance of these is consistent with peritoneal bands.

Cystic Duct

The course and caliber of the cystic duct, hepatic ducts and the common ducts are normal. Examination shows no evidence of stone.

Following the fatty meal, there was a satisfactory contraction of the gall bladder. Impressions: (1) Excellent liver function and gall bladder concentration power with early and adequate opacification. (2) Extrinsic bands across the gall bladder and the neck of the junction of gall bladder and cystic duct as described above, no evidence of stones. (3) Normal contraction of the fatty meal.

Following the roentgenological examination, the pains were less severe, however, the patient developed chills; his temperature which had been normal on admission, rose to 100.2F. Anorexia was marked. The patient would not eat or drink. He was given I.V. fluids and the laboratory report showed the WBC reaction typical of an inflammatory process (16,500). Antibiotic therapy was started, with rapid effect. There remained the localized gall bladder tenderness; and due to the normal gall bladder function the possibilities of a peptic ulcer was given consideration. There was no abdominal distention, no abdominal discomfort, no history of melena or hematemesis. Five days after admission the gall bladder region remained tender, the temperature was normal, and laparotomy was decided upon. The following were the surgical findings: Upon opening the abdomen through a transverse subcostal incision, the gall bladder was covered with a thick fibrinous exudate. This was removed from the gall bladder which was normal. Extending across the ampulla and cystic duct, from the region of the first portion of the duodenum, was an organized fibrinous band of exudate. This was removed. Examination of the duodenum and pylorus revealed that the fibrinous material was originating from the region of the sealed-off duodenal perforation. The duodenum and pylorus in this area were markedly thickened and edematous.

The fibrous exudate over the perforation was not disturbed as the opening appeared to be thoroughly sealed off. The abdomen was explored for gastric content in the right gutter. No evidence of abdominal contamination was found. The abdomen was then closed without drainage. The post-operative care following recovery from the anesthesia was that described in the management of the other ulcer patients. Roentgen examination four days after surgery revealed the following: The patient's recent surgery precludes examination with intra-abdominal increased pressure and extensive pressure spot films. There was no abnormality of the swallowing function, no identified hiatus hernia, varices or cardio spasm.

Gastric Mucosa

The gastric mucosa was relatively normal in appearance, there was no marked hypersecretion.

Peristalsis was promptly initiated and moved the contrast medium through a small but adequate pylorus. There was a barium density seen in a number of projections approximately 5 mm in diameter, occasionally smaller, surrounded by a zone of edema, compatible in appearance with an acute, shallow peptic ulcer.

Minimal hypertrophy of the duodenal loop was noted. At three hours, the stomach was empty. The bulk of the barium was in the terminal ileum, a normal transit time. There was no gross abnormality of proximal small bowel. Impressions: (1) Acute duodenal ulcer at the base of the duodenal bulb. (2) No other significant abnormality.

The C-Cs treatment was started on the day of

surgery and continued for ten days with symptomologic healing. On 10-5-59 the follow-up X-ray report was:

The patient was recovered from his recent surgery permitting examination in the deep Trendelenburg position. This disclosed no abnormality of the esophagus. There was no marked hypertrophy of the gastric mucosa, there were no retained secretions at the beginning of the examination.

Peristalsis moved normally through the body of the stomach, there was no evident gastric ulceration. There was no tenderness to palpation over the duodenal bulb, no spasticity. Examination of the region of the previously noted ulcer in multiple projections revealed no indication of this defect. In comparison with previous studies the mucosa of the duodenal loop was much more normal in appearance.

Barium Retained

At three hours there was minimal retained barium mixed with food, a finding not to be considered abnormal in this patient. The majority of the barium had moved in a smooth column into the terminal ileum and ascending colon, a normal transit time, a normal three-hour appearance. Impression: (1) Healed duodenal ulcer; no current abnormality of the upper GI series.

Case 10, H.H.G., Hospital #63,847 male, age 54, admitted to the hospital on 9-28-59 with a C.C. of inability to retain any foods or liquids, accompanied by vague upper abdominal discomfort. Onset about eight days ago, with hematemesis six days ago and followed by tarry stools. His past history was that of indigestion over the past few years, relieved with gelusil, aludrox, etc. About one year ago he was treated for a duodenal ulcer. Physical examination revealed slight tenderness of the epigastrium. All systems were negative for pathology. The clinical laboratory reported W.B.C. 10,800, Hb 88%, Hematocrit 43%, Hemoglobin 13.75 grams. Roentgenological examination on 9-28-59 revealed there was no abnormality of the swallowing function except a minimal slowness. There was no indication of cardio-spasm or of esophageal varices or hiatus hernia. There was no fluid retention at the beginning of the examination of the stomach. The patient was kept under intermittent observation for a period in excess of three hours. During the interval, active peristalsis was never observed. While at rest a small quantity of barium filtered through the pylorus into the small bowel. A minimal non-specific abnormality of the appearance of the jejunum was noted. The stomach was high and transverse, inaccessible to direct palpation.

Examination with conventional techniques and with air contrast methods were attempted. The pylorus remained tightly closed. In the superior-posterior aspects of the duodenal bulb there was a persistent barium fleck seen on many of the films which may represent a psuedo-diverticulum of a peptic ulcer. The barium suspension was markedly flocculated by hyperexcretion and this, together with a lack of peristalsis, obscured the fine mucosal detail. Impression: Almost complete atony of the stomach, marked by pylorospasm with markedly delayed emptying of the stomach. Persistent irregularities of the appearance of the duodenal bulb suggesting an acute peptic ulcer. An ideal demonstration of an ulcer crater cannot be accomplished under these conditions. Reexamination after a suitable period of medical management was indicated.

Reexamination

The patient was then started upon the treatment as outlined in Chart 2, and reexamination was then done. The following is the report: 9-30-59 additional films of the duodenal bulb were made. In comparison with the previous films of 9-28-59, there was no pylorospasm. Peristalsis moved smoothly through the body of the stomach. The duodenal bulb was deformed with a persistent barium density in the pyloric canal compatible with a psuedo-diverticulum of a chronic duodenal ulcer. Spasm of the bulb distal to this area was noted, there was no marked abnormality of the mucosa of the duodenal loop beyond the bulb. Impression: Pseudo-diverticulum of the penetrating duodenal ulcer within or immediately adjacent to the pyloric canal, marked secondary persistent spasm, no further evidence of functional obstruction. The Cosa-Signemycin/chymotrypsin treatment, as described, was continued for ten days, and on 10-13-59 upper G-I recheck films were obtained, with the following findings reported: There was no abnormality of the swallowing function, no cardiaspasm or evidence of

esophageal varices. On this examination the patient was asymptomatic, he was placed in the very steep Trendelenburg position and the distal esophagus more adequately examined than possible on previous studies. There was a small herniation of the gastric mucosa apparently of the short esophagus, which could be demonstrated only with increased intra-abdominal pressure.

A contraction ring was present at the junction of the esophageal and gastric mucosa but there was no evidence of active peptic ulceration. Demonstration of this abnormality did not cause any complaints. There was no fluid retention in the stomach, the gastric mucosa was now normal in appearance. Peristalsis began immediately, delivering adequate quantities of contrast medium through a normally patent pylorus. There was no tenderness to palpation, no current spasm of the duodenal bulb. This structure was examined in multiple projections and a barium-containing defect was not identified in this region of the previous ulcer. There was no residual scarring demonstrated. At three hours, minimal barium remained mixed with food in the distal stomach. The remainder of the barium column was in the terminal ilium and ascending colon, an essential normal transit time. The proximal small bowel mucosa was then normal. Impression: In the deep Trendelenburg position there was an apparent hiatus hernia of the short esophagus with a transient contraction ring of the esophageal and gastric mucosa, suggesting old scarring, no evidence of current acute inflammatory disease. No indication of symptoms during the demonstration of this abnormality. No mucosal abnormality of the stomach, active peristalsis, no fluid retention. No spasm, tenderness or deformity of the duoden bulb, no peptic ulcer demonstrated.

The patients were then placed on a diet of all foods excluding alcoholic beverages and acid-type condiments, etc.

Summary

Proteolytic enzymes are highly specific in their action (10). In consideration of their chemical structure and biological activity, it has been shown that their action is one of absolute specificity (3). It has been known that chymotrypsin has specific peptidase and esterase activities (11), also that there are natural occurring trypsin inhibitors against chymotrypsin (22). This inhibitor of chymotrypsin may be found in various animal species (5), and the opposite is true for chymotrypsin, which may be obtained from mammalian pancreas, and has been found to be an inhibitor of the gastric secretions.

In the classification of proteolytic enzymes, the chymotrypsin action as an inhibitor of gastric secretion also has been shown to produce changes in the duodenal mucus (20), thus inhibiting or neutralizing hydrochloric acid and simultaneously eliminating the peptic activity of the gastric secretions thereby permitting healing of the peptic ulcer.

Ten patients were treated for an acute phase of a peptic ulcer, with this proteolytic enzyme obtained from mammalian pancreas (chymotrypsin). This enzyme produced effective neutralization by inhibition of hydrochloric acid secretion with elimination of the peptic activity. With the combined use of Cosa-Signemycin to control the cellulitis of the ulcer process, healing of the ulcer process was obtained in less than two weeks, without the use of antiacids, anticholinergic drugs or the usual ulcer regime.

Conclusion

The results of this study indicate that the combined administration of Cosa-Signemycin and chymotrypsin in the treatment of gastric or duodenal ulcers confers sufficient therapeutic effect to arrest and heal the disease. Although some response may be obtained from the use of chymotrypsin alone, an excellent response was obtained in all patients with an acute peptic ulcer by the combined use of oral Cosa-Signemycin with the intramuscular injections of chymotrypsin. Follow-up X-ray examination revealed complete healing in the treated cases.

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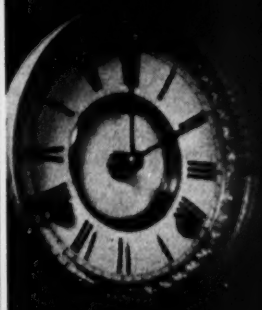
Comment

After SOUTHWESTERN MEDICINE had gone to press for its February 1960 issue, Dr. Samuel L. Cohen of Phoenix, whose article, "An Axial View of the Hip Joints," was being published in the February issue of SOUTHWESTERN MEDICINE, learned that the view expressed in his article was originally discovered in France in 1920 and has been utilized to a considerable extent by various orthopaedic surgeons and radiologists.

Dr. Cohen expressed regret that he was unable to inform SOUTHWESTERN MEDICINE of the origin of his view in time for mention to be carried in the bibliography accompanying his article.



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R. E. Thomason General Hospital, El Paso

December 17, 1959 — *Case No. 1341*

F. P. Bornstein, M.D., *Editor*

Presentation of Case by Charles Logsdon, M.D.

History: Dr. Nathan Kleban:

A 33-year-old Latin-American man was admitted to the hospital on September 28 and died on October 5, 1959.

At age 14 the patient had epistaxis, arthritis in both knees and chest pain. When seen in cardiac clinic at age 15 a loud systolic murmer and a soft diastolic murmer were described at the apex. Blood pressure was 110/50. There was no clear-cut heart failure. Only a systolic murmur was subsequently described over a two months period. He was rejected for military service because of a heart murmur.

Cardiac decompensation became manifest in 1951 at age 25. Rhythm was atrial fibrillation. Later attempts to convert to normal sinus rhythm with quinidine were unsuccessful. Reported were grade IV systolic murmur and duplication of the second sound at the apex, diastolic murmur at the base and elevation of the lower end of the sternum in systole. It was the impression of the cardiologist that there was recurrence of active rheumatic carditis, mitral stenosis and insufficiency, aortic and tricuspid insufficiency.

Numerous hospitalizations occurred because of symptoms and signs of cardiac decompensation. In 1954 there was transient loss of consciousness thought to be due to possible cerebral embolus. Pericarditis was a diagnosis in 1955. In that year cardiac surgery was considered but not carried out. Sulfa and benzathine penicillin were used prophylactically but irregularly. Epistaxis and ecchymoses occurred in 1958. He worked as a boiler maker, truck driver and grocery clerk until 1958.

An anticoagulant, warparin, was administered for 10 days in August, 1959, for transient neurological changes.

Cardiac catheterization was done at another hospital on September 19, 1959. Blood pressure was 130/40. Impulses were felt at the left sternal border in the fifth intercostal space and in the eighth at the mid-axillary line. First mitral sound was loud. A high pitched grade II-III decrescendo murmur was heard at the base and transmitted to the apex. There were no thrills, clubbing, cyanosis or edema. Peripheral arterial pulses were strong. Rales were heard over the left lower lobe. The liver was enlarged. Electrocardiogram suggested left ventricular hypertrophy.

On X-ray film and fluoroscopic examination of the heart there was considerable enlargement of both ventricles and particularly of the left atrium. Effusion and hilar dance were absent. Catheter diagnoses were: Rheumatic heart disease; aortic regurgitation; pulmonary hypertension and pulmonary vascular disease; congestive failure.

Final hospital admission occurred on September 28 because of sudden onset of right hemiparesis and inability to speak.

Physical Examination:

T. 99, P. 56 (radial), R. 28, B. P. 120/50.

The patient appeared semi-conscious, was unresponsive. There was right hemiparesis. Cardiac findings were unchanged. There was a slow ventricular rate with atrial fibrillation. Liver was enlarged but there was no edema.

Hospital Course:

Opening pressure during lumbar puncture was equivalent to 110 m.m. of water. After about 15 cc. of clear fluid was removed pressure was 65 mm.

Heparin, warparin, digitalis and quinidine were prescribed. There was no remarkable worsening of the chronic congestive heart failure. Despite some improvement in state of consciousness death occurred quietly on October 5, 1959.

Laboratory Findings:

Blood Counts: 9-28-59—Hb. 16.3 gm., Ht. 48%, WBC 9,200, Stabs. 6, Segs. 78, Lymphs. 14, Monos. 2.

Prothrombin time: 9-28-59—52% (Pt. 17″, C. 13″). 9-29-59—28% (Normal Proth. time 13 sec., Pt's 19 sec.). 9-30-59—27% (Normal Proth. time 13 sec., Pt's 19.5 sec.). 9-30-59—17% (Pt's 25.5″, C. 13″). 10-1-59—16% (Patient 27″, C. 13″). 10-2-59—13.5% (Pt. 31″, C. 13″). 10-3-59—13.5% (Pt. 27.5″, C. 13″). 10-4-59—27% (Pt. 19.5 sec. control 13 sec.)

Urinalysis: 9-28-59—Amber, turbid. acid—S. G. QNS, albumin 4+, sugar negative, WBC 2-3/hpf, RBC 4-8-hpf, occasional hyaline casts, few bacilli.

Spinal fluid: 9-28-59—WBC 3, polys 2, lymphs. 1, Sugar 71 mg.%, total protein 26.6 mg.%. Gold curve 0011000000.

Blood chemistry: 9-28-59—CO_2 capacity—19 mEq/L. Urea nitrogen—28.2.

Serology: 9-28-59—negative

Blood culture: 9-28-59—no growth. 9-29-59—no growth. 10-1-59—no growth.

X-ray chest: 9-28-59—Portable re-examination of the chest and comparison with previous study reveals a generalized intensification consistent with accompanying congestive failure. The previous observed area of pneumonitis is disappeared. Conclusion: Rheumatic heart disease with accompanying congestive failure.

X-ray Discussion: Dr. Vincent Ravel:

We have here a series of four sagittal views of the chest, the first one was taken in 1951. Over a period of approximately eight years there has really been very little change except in the last film, where we have signs of congestive failure. There is marked enlargement of the left atrium; the pulmonary artery segment is quite prominent; the right atrium is enlarged as well as the left ventricle; and this brings to mind the possibility of a double lesion, a mitral and aortic lesion, together with a possible congenital anomaly, such as a septal defect.

Clinical Discussion: Dr. Charles Logsdon:

I first saw this patient in 1951 at which time he was in congestive failure. He was 25 years old. A diagnosis of mitral regurgitation as the principal lesion, of mitral stenosis, and a very significant aortic regurgitation, a relative tricuspid regurgitation and of uncontrolled atrial fibrillation was made. Since we know that these patients are quite capable of living for a long time with severe valvular lesions, as he subsequently demonstrated, a diagnosis of rheumatic myocarditis was also made at that time to explain the severe congestive failure.

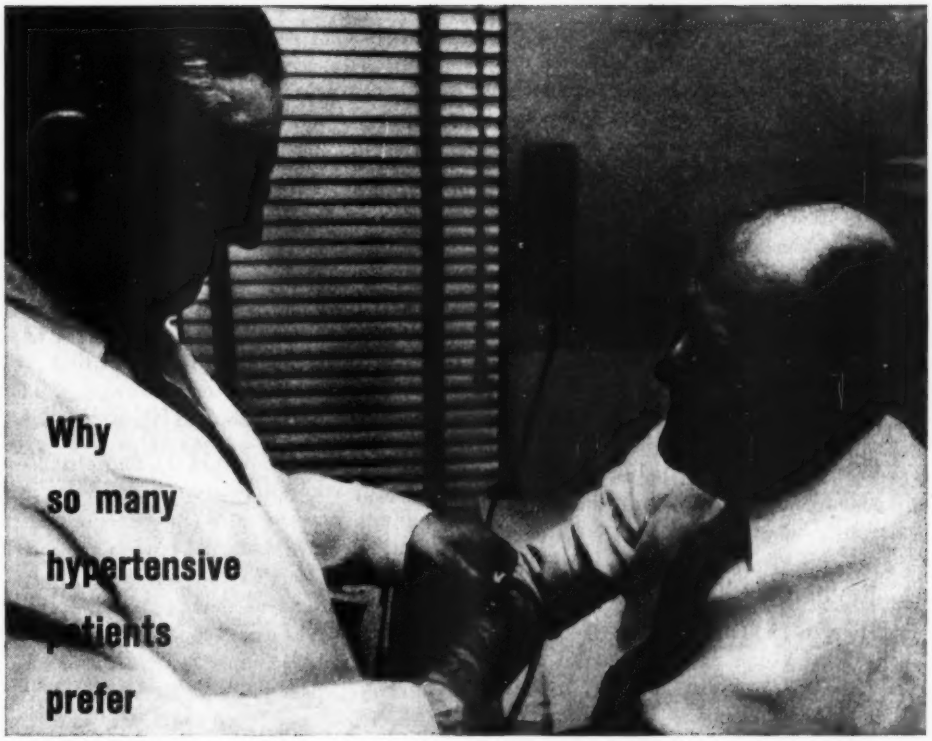
It was noted in the protocol that the inferior portion of the sternum was visibly elevated in systole which in this type of case is presumptive evidence of tricuspid insufficiency as it implies a very considerable enlargement of the right heart. This also implies a dilatation of the tricuspid ring with relative insufficiency of the tricuspid valve.

For several years after we saw this patient the first time, he had off and on a very frankly pulsating liver. In this phase of their disease these patients usually die. They usually die of progressive, intractable heart failure or a complication of their heart failure or heart disease such as embolism. Surprisingly, this patient showed a good deal of recovery over a period of years. His heart remained large, his fibrillations continued and his valvular lesions were apparently unaltered. Nevertheless, he got better. His liver shrank in size; it was no longer palpable; his sternum was not elevated in systole; and he did reasonably well. His treatment was sporadic. He would go to California and stay for a period of time and he would show up in the clinic, undigitalized, and in failure. Finally in 1956 he had married for a second time, I think he stayed digitalized until his death.

At that time, in spite of his large heart and all of his valvular lesions, he was in very good condition. In April of 1956 he was approved for vocational rehabilitation and he took a course in barbering. He came back in a few months and for three years or more, made his living as a barber and did fairly well. He worked most of the time and supported the family which he started in those three years.

The patient had heard a good deal about cardiac surgery over a period of years, and he had always said if an operation was ever devised for his type of heart disease, he wanted to be a guinea pig.

In August of 1959 I saw the patient before he came into the hospital. I saw him in the office because it was not a clinic day and he was feeling very bad. He came into the office. His heart was fast, although he was taking digitalis; and his liver was pulsating again, which was the first time in some years he had demonstrated that phenomenon. That was his complaint, by the way, that his abdomen was very uncomfortable because of a



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*Herrmann, G. R., Vogelpohl, E. B., Hejtmancik, M. R., and Wright, J. C.: J.A.M.A. 169:1609 (April 4) 1959.

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large pulsating liver. It was shortly after this, a matter of days, that he entered the hospital with the transient neurological changes referred to in the protocol, which were thought to be due to a cerebral embolism.

On this hospitalization he ran a low-grade temperature, rather more than is usually found in a patient with slight cerebral embolus and nothing more; although I presume he had sufficient congestive phenomena in various organs to account for the temperature, which I believe went over 100 on occasion, perhaps every day while he was in. At any rate, at that time I wondered if his general condition might be masking something like an S.B.E. which seemed very unlikely. After this he was seen in the out-patient department. He was somewhat better, but he didn't compensate as he had on other occasions, and he was quite insistent about surgery.

Well, it seemed highly unlikely that he could be submitted to any kind of cardiac surgery; but I did send him to Dr. Crossett and asked him to reconsider him, and some days later he was admitted to the hospital for cardiac catheterization. He was seen after that and he was in fairly good condition, and then nine days after the catheterization he again suffered a cerebral episode of some sort and was admitted to the hospital semi-conscious and with hemiparesis, and died on the seventh hospital day.

The cardiac catheterization of course accomplished nothing except for some interesting data. It is difficult to see how it might have contributed in any way to his terminal illness. Once this type of patient starts to throw emboli, he approaches the terminal phase of the illness. Once they start throwing emboli, they don't stop because the conditions which produce the emboli and make them mobile, go right on. You can't alter them; and so the first embolus is usually a warning of what's coming up.

I discussed with Dr. Crossett the possibility of operating him, and I am sure he didn't take it too seriously; but I think the only thing that might have been accomplished in this boy, assuming that a valvular repair was impossible, would have been a tie-off of the left auricle, which might have slowed down his embolization and might have given him, had his heart compensated, another year or two. It was my impression that his principal lesion was mitral and the valve was largely regurgitant and secondarily stenotic, and that the next most important lesion was aortic regurgitation.

These two lesions have their principal regurgitation of course on the left ventricle and the left atrium, which becomes quite huge as you can see here, it contributes to that double shadow, that shadow that you can see on the right and there is another one that you can see on the left. Of course the left atrium becomes the repository for a great many thrombi, which under certain conditions will start moving, and with a rise in venous pressure in the pulmonary circuit and with the subsequent rise in the arterial pressure, the right side of the heart begins to enlarge.

We see these patients in the state that he was in and with regurgitant pulmonary valve and regurgitant tricuspid valve and a generalized cardiomegaly. Once that starts, recovery for any length of time is not very likely, even though it did occur in this boy, and even though Dr. Crossett has been using an operation for repair of this type of lesion, it's very doubtful that a patient of this type would even pass through the first stage of anesthesia without a cardiac arrest, and certainly these old rheumatic myocardia are very hard to get started again when they do stop. If someone wants to discuss the catherterization data I would be glad to have them do so, although I dare say it is not too pertinent to the remainder of the discussion.

About all there is left to comment on is the mode of death in this patient. It seems to be a fairly straightforward sort of case. The usual mode of death is cerebral embolism, and it is most certainly the most attractive diagnosis, not only in this case but in all such cases.

There are a few things about mitral thrombi and their mobilization that are rather pertinent. Autopsied cases like this usually show numerous emboli, and for the most part they are old, in the lungs, in the spleen and the kidneys. Most of them, because of the many symptoms, and the morbidity of the illness, pass unnoticed. In almost all cases the abdominal emboli cause pain and fever, and sometimes they even cause abdominal operations. One such case of mine was operated, for an appendiceal abscess, by some very good men, who never found the abscess.

If the emboli end up in the lungs you can see, toward the end anyway, how difficult it would

be for them to cause symptoms that would be remarkable. After all, it is not unusual for these patients to spit blood, not unusual to have pulmonary congestion, so the pulmonary emboli frequently pass unnoticed, although the X-ray report on the protocol would indicate that he perhaps did have a pulmonary embolus or maybe pneumonia.

This type of patient does not as a rule start the formation of thrombi in the atrium until congestive failure and atrial fibrillation intervene. That is true whether it is a stenotic lesion or a regurgitant lesion, although in the stenotic lesion it would seem, statistically, the formation of thrombi is more likely, and occurs more readily than in the regurgitant lesion; but once congestive failure and atrial fibrillation have supervened, then thrombus formation is inevitable.

One of the hazards, and we see this quite often, of bringing a patient out of atrial fibrillation and congestive failure, is that in doing so the circulation is improved and thrombi which have formed are mobilized and go to the periphery, either cerebral emboli or pedal emboli, or more likely, to spleen and kidney. This patient's first cerebral episode occurred after I saw him in the office and increased his digitalis and gave him a diuretic.

I might well have caused the first episode by improving his circulation somewhat. That of course is the same hazard that exists in converting a patient from atrial fibrillation to sinus rythm, with the added hazard of a functioning atrial myocardium, and it would be mentioned that in almost all of these cases we see a recurrence of myocarditis and frequently endocarditis. The endocardial lesions themselves cause small inflammatory vegetations on the valves which can be the source of small emboli. Another source of emboli in these cases is S.B.E. Bacterial vegetations on the mitral or aortic valve could cause this patient's terminal illness; but with the many findings that he had such a lesion would probably go undiagnosed. A patient who has had as much congestive failure as this patient had over a period of time, does not as a rule develop S.B.E.

Other mechanisms that could have produced the terminal illness — I won't go into anything that isn't vascular — are, asystole, produced either by over-digitalization, with heart block and Adams-Stokes syndrome, could produce cerebral episodes particularly like the first one, but it is difficult to see how it could have accounted for the terminal episode. Also, one thing that is rather far-fetched, but perhaps should be mentioned, is that he could have had a giant thrombus in the left atrium which would intermittently occlude the mitral orifice. This is sometimes diagnosed ante-mortem and on occasion is associated with a definite cerebral lesion with hemiplegia.

In summary, it is my opinion that this patient had a rather fortunate evolution of his disease and that he ultimately succumbed to repeated cerebral emboli.

Dr. Saul Appel:

The cardiac catheterization was done when the patient was in relatively good shape. We had seen him a week before and his heart rate was well controlled by the time catheterization was performed. He was adequately digitalized. There was no definite evidence of tricuspid insufficiency, although the pressures in the right atrium and right ventricle were elevated, specially the end diastolic pressure, which indicates that he was in left sided heart failure.

Now the data that Dr. Verosky calculated out on the basis of right heart and left heart catheterization, were slightly affected by technical factors, that is, damping. However, there was very good evidence that the man had a significant aortic insufficiency, with, we weren't able to prove hemadynamically, significant aortic stenosis. His pulmonary vascular resistance was definitely elevated, 700 dyne seconds cm. whereas the normal is anywhere from 50 to 250. That is certainly elevated, although not as high as some of the chronic rheumatics that we have seen.

The cardiac output was greatly diminished. He had an output of 1.6 liters per minute per liter square, whereas the normal is about twice as much; and his systemic arterial venous oxygen difference was greatly elevated, 10 vol. %, instead of a normal which is half as much, indicating a marked congestion in the periphery with peripheral extraction of oxygen being very high, as in most cases of a low out-put heart failure.

As Dr. Logsdon said, the catheterization data wouldn't make very much difference in this man's final story, because he was a very severe chronic cardiac, he had been in failure most of the time the last eight years of his life, eight out of 33 years, and he had suffered a probable cerebral

embolus in 1954 and another one in August of 1959 just a month before he was catheterized. He was on anticoagulants after the second cerebral embolus.

However, in patients like this who are class IV cardiacs and for whom the prognosis is hopeless, we feel that it is worth catheterizing them if there is any chance they might improve and come to surgery. It is true that the risk for surgery in such cases is terribly high. On the other hand, when one makes such complete physiological study, one obtains a very strong opinion about the prognosis.

In addition, we have a patient here who said he was willing to be a guinea pig, which of course we wouldn't seriously entertain; but if we have a patient with the attitude that if anything can be done he is willing to go through with it, then you are willing to try and do the best you can. We don't feel that the catheterization had anything directly to do with his demise.

It is true that during the procedure, if there are thrombi, one might possibly dislodge a thrombus and form another embolus, but that should have been evident within the period of catheterization, or next day. In a man who has at least two emboli, as Charlie said, chances are that he had a lot more that were never recognized because they never hit a structure like the brain.

There is still one possibility that may have increased the likelihood of a terminal embolus; when he left the hospital after the admission in August before the catheterization, he was put on anticoagulants. He was on 5 mg. of coumadin a day from the 10th to the 21st of August, which is within the therapeutic range.

When he left the hospital he was given no more coumadin because I saw him on the 11th of September, only three weeks later. He told me then that he was given medicine to thin his blood. I therefore asked him to bring in all his pills, and among them there was no anticoagulants. He had not been back to the cardiac clinic since his discharge three weeks before. Therefore, we might have deferred a catheterization until his anticoagulant therapy was resumed.

This problem comes up very often in treating patients who are on prolonged anticoagulants. What are you going to do when they have to have a dental extraction or surgery? We hospitalize them, discontinue the anticoagulant, give them a little vitamin K if something is urgent, and has to be done in the next day or so, and then following the surgery or the dental extraction, resume the anticoagulants.

The lapse of time there is approximately a week or less and in the experience of most people there is no increased incidence of thrombosis or embolization if the period off the drug is only a week or two. When the period the anticoagulant is stopped is for four weeks or five weeks or six weeks, then there is an increased tendency to thrombosis.

I agree entirely with everything Charlie has said about the course of rheumatic heart disease. Here is a man who exhibits it very well, the chronicity of the patient with mitral disease and an aortic lesion which was not his chief lesion. It is true that with aortic disease, and this is mainly a hemodynamic lesion, the course is much more rapid until death.

Dr. E. S. Crossett:

One of the questions that came up about cardiac catheterizations is the indications for doing them. I agree with Dr. Logsdon that the diagnosis in this case is perfectly apparent. From a practical standpoint there was little reason for doing a cardiac catheterization. The only reason that I personally want to do catheterizations in patients who are remotely or actually being considered for surgery on the left side of the heart, is to measure their pulmonary resistance.

I think that this business of pulmonary resistance is something that the average doctor does not hear about. It is very important in the surgical treatment of congenital or acquired heart disease.

If the lungs are congested from overflow from either ventricular or atrial septal defect, or anomalous pulmonary drainage, where more blood than normal flows through the lungs as a result of these anomalies, or if the lungs are congested from backing up of blood as a result of mitral valvular disease, then certain changes over a period of time will take place in certain people's lungs.

These changes consist first of spasm of the percapillary arterioles in the lungs, and then as time goes on, in certain individuals, but not all individuals, — for some reason it is just a selective disease — these precapillary arterioles not only have spasm but they actually have organic thickening of the walls and thickening of the intima so that they become organically obstructed to the flow of blood.

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Now this is a protective mechanism of the lungs to prevent the capillary beds from being congested with blood. I have no proof that it is the same mechanism in acquired and congenital heart disease that causes these changes, but I think that it does. It just prevents blood from congesting the capillaries and entering into the alveoli.

Well, it is important for the surgeon to know before the surgery, whether or not the changes in the precapillary arterioles are on the basis of spasm, which is reversible, or on the basis of actual irreversible organic changes, because, if it is on the basis of organic changes, then correcting the heart lesion will not cure the patient.

He still has an obstruction to the flow of blood in his lungs. Most of them die during surgery, and the ones that don't die are helped very little by the surgery.

Status of Arterioles

In order to determine the status of the arterioles in the lungs, if you will put a catheter into the pulmonary artery, measure the pressure, find it is extremely high, probably equal to systemic pressure, then let the patient breathe oxygen for a period of five to ten minutes, we use ten minutes, and then remeasure the pressure. As a rule if the obstruction is due to spasm, the pressure in the pulmonary artery will drop to some degree.

If it is an organic change that is irreversible, it will not drop. Actually, in the patients that we have had the pressure has gone up after breathing oxygen, and every one of those except one has died when we operated. That is the only reason for doing a cardiac catheterization on this patient.

As far as the data obtained from the left heart and so on, it's interesting, but from the practical standpoint, at least at the present status of our knowledge, I see no value in it. But I think that any time you can collect data and make a record of it you ought to do it, because some day it may be of some value to you if you look back through your cases.

Cardiac Surgery

Now then the indications for cardiac surgery in a patient who is severely ill with acquired valvular disease: so far we have not operated on any aortic insufficiencies. The reason is that no one has devised a really good operation. A few procedures in real desperate cases I think are worth a try, such as removing one of the valve cusps of the aortic valve and making a bicuspid valve out of it. Another is to sew a piece of Ivalon sponge onto one of the valves so it occludes the opening and perhaps will prevent regurgitation. But these are desperate procedures, and I think they should be reserved for those real far advanced dying cases at the present time just as a last hope for them.

Now as far as your mitral stenosis and insufficiency, we all know that mitral stenosis is very well treated by surgery. Our results are good and it is a worthwhile operation. Where there is stenosis and insufficiency combined, and there is a group of cases in which there is only insufficiency. We have done about 11 procedures on acquired valve disease — nine procedures, on the left side of the heart, 11 procedures including two congenitals, but in mitral insufficiency, the results are not good, according to people around the country.

We have not been disappointed too greatly. Some of them have residual murmers, some of them are hard to evaluate as far as symptoms are concerned. The first case that we did, for instance, was a woman who had had ascites for a year, she had had three episodes of ascites, a big liver, ankle edema and so on. We operated on her and put an Ivalon prosthesis in where her posterior valve had been destroyed by rheumatic fever.

Post-operatively she had no murmur, she survived the procedure without any trouble except that she had an air embolus which caused cerebral symptoms that cleared up in three or four days. She became able to take care of a family of three or four children, doing so without symptoms.

She now has a systolic murmur, about a grade II systolic, but no cardiomegaly, no enlarged liver, and is a perfectly happy woman. If you can get results like that, treatment of mitral insufficiency is worth a try, and we usually take the pretty far advanced cases. There are others which have been improved but still have residual murmurs and so on, so you can't conclude that this treatment is very good, it is just worth a try.

Dr. Logsdon mentioned the fact that a seriously ill patient like this will die of a cardiac arrest often from the anesthesia. It is absolutely true, we have had two deaths and both of them died right after the anesthetic was given, from a cardiac arrest and we were never able to resuscitate their hearts and you can't blame anything for

that except poor selection of patients, and with experience I think we will begin to select our patients a little better.

Cause of Death

Now as to the cause of death in this patient, I think that possibly a needle was put through his left auricle to measure his left auricular pressure and may have precipitated a clot. The other possibility to me is a sub-acute bacterial endocarditis with a vegetation breaking off. My first guess is that he had S.B.E. as well as these other things and a vegetation broke off, but I can only guess about it.

One of the things that was brought up was tying off the atrial appendage to prevent clots. The only death that I have had from a mitral stenosis, which was a closed mitral stenosis operation in El Paso, was a patient who had had emboli so that he had a gangrenous leg. He was unconscious when I operated on him. Post-operatively about his sixth or seventh day he developed a thrombus where I tied off the appendage. It plugged up his mitral valve and killed him. I really don't think that tying off the atrial appendage is much value, particularly in a case where you can't do anything but tie off the appendage.

Dr. Logsdon:

I would like to say two things about anticoagulant therapy in this type of patient. I wouldn't argue too much about it because much of it is a matter of opinion. One, a patient of this kind with congestion of a good many organs, with chronic liver disease, is a patient very difficult to control on anticoagulant therapy, and controlling him here at the out-patient department is almost impossible. Anticoagulant therapy is something that a man has to be on every day and I can't control patients in my cardiac clinic because I don't see them but once a week.

In the second place, it may not even be a good procedure at this stage of the game. In fact, I think it is a definitely dangerous procedure. If a patient is anticoagulated and throws an embolus, wherever he throws it he is going to get in a great deal of trouble because of hemorrhage. Once a patient has thrown an embolus from his left atrium, we have no idea how many more he has or when they'll blow.

It seems to me that if we are going to start anticoagulant therapy on these people it should be started early, way back in 1942. We couldn't have in 1951 because he was even more ill then than now, when he died. Some cardiologists put their patients on anticoagulant therapy when they go into atrial fibrillation. That might be a safe procedure, but after they have started throwing emboli it is exceedingly dangerous. It will convert small emboli phenomena into very tragic ones in a hurry.

Clinical Diagnosis: Rheumatic Fever

Dr. Logsdon's Diagnosis: Repeated cerebral emboli, mitral insufficiency.

Pathological Diagnosis: 1. Mitral insufficiency, rheumatic type, of extremely long standing, associated with relative insufficiency of all valves. 2. Extreme cardiac hypertrophy and dilatation. 3. Myocardial scarring in apex. 4. Acute rheumatic myocarditis. 5. Acute encephalomalacia, probably embolic.

Pathological Discussion: Dr. Bornstein:

The external examination of this body was not remarkable. The main findings were in the thorax. The pleural cavities, amazingly enough, were free of fluid and adhesions. The pericardial sac was tremendously dilated, the apex being at the eighth rib in the lateral clavicular line. The pericardial sac was completely obliterated.

There was no gross evidence of any damage or injury from the preceding catheterization. The heart was markedly hypertrophied and weighed 750 grams. The myocardium was reddish-brown and firm except in a thinned out area in the apex of the left ventricle. The myocardium in the right side measured 12 mm. This represents hypertrophy of the right ventricle and 22 mm. on the left side, which is not too much of a hypertrophy.

The valves of the right side of the heart were anatomically normal but functionally incompetent. The heart was enormous in size, larger ever than would appear from the weight, and this was due to the enormous enlargement of both atria which took up about 50 percent of the size of the heart.

Main Lesion

The main lesion was one of the mitral valve. The mitral valve (Fig. 1) measured 10 cm. across. On inspection they showed total fusion of the valve leaflets, forming nearly immobile valve ring

with obvious insufficiency. On inspecting the inferior surface of the mitral valve (Fig. 2) one saw marked shortening of the papillary muscles and thickening of the chordae tendineae. The aortic valve measured seven cm. across and showed some thickening of the cusps but was essentially anatomically normal. Examination of the artia failed to reveal any mural thrombi. However, a few mural thrombi were attached to the endocardium of the left ventricle in the region of the thinning out previously mentioned.

There were numerous old infarct scars in the spleen and both kidneys. The brain showed softening of the left hemisphere, with foci of xanthochromic discoloration and sectioning showed numerous hemorrhagic foci in the cortex suggestive of recent infarction.

Microscopic examination in addition revealed mild pulmonary edema and some bronchopneumonia. The microscopic examination of the heart revealed scarring and foci of myocarditis including Aschoff bodies.

Several questions arise in interpreting this lesion. Basically this is a pure mitral valve lesion. The heart became decompensated and mural thrombi were formed, cerebral embolization, coma and bronchopneumonia ensued which produced death.

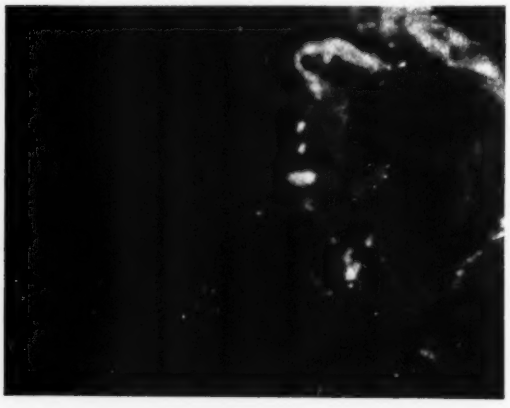

Figure 1

Role of Myocarditis

It is more difficult to evaluate the role of the myocarditis. It is true that patients with valvular lesions who have an even course on digitalization and who then suddenly decompensate show large areas of acute rheumatic myocarditis. On the other hand, since cardiac surgery has been practiced on a larger scale, it has been common to find small foci of active rheumatic inflammation in the auricular appendages. It therefore becomes questionable how much importance one should attach to the presence of active rheumatic foci in interpreting the course of the disease.

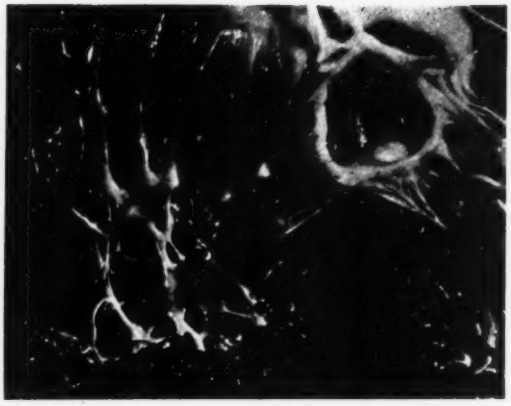

Figure 2

The mural thrombi at the thinned out area are obviously secondary in type, the thinning out being due to previous emboli entering the coronary arteries. The case is unusual inasmuch as it presents a pure mitral insufficiency with all other changes being secondary to this lesion. It is also the first time that we have the opportunity to discuss a case with complete catheterization and evaluate a mitral heart not only as a medical, but also as a surgical problem.

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